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## The association of intra-abdominal infection and abdominal wound dehiscence / Discussion

*The American Surgeon*; Atlanta; Jul 1998; [Debra J Graham](#); [Jean T Stevenson](#); [Christopher R McHenry](#); [Charles Lucas](#); [Homer M Smathers](#);

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### Abstract:

*A study of abdominal wound dehiscence was conducted to determine the incidence and to identify prognostic factors for associated intra-abdominal infection. Lucas and Smathers comment on the study, and Graham responds.*

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#### [Headnote]

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#### [Headnote]

Concurrent infection is a risk factor for abdominal wound dehiscence. We reviewed our experience with fascial dehiscence to determine the incidence and to identify prognostic factors for associated intra-abdominal infection. Over a 7-year period, 107 patients with abdominal wound dehiscence were identified. Seventeen were managed nonoperatively, and 90 underwent exploratory laparotomy, 43 of whom had no intra-abdominal pathology and 47 of whom had intra-abdominal infections. Demographic factors, comorbid diseases, and potential indicators of systemic infection did not distinguish patients with intra-abdominal infection from those without. Patients with an intra-abdominal infection were more likely to have undergone an emergency operation (74% vs 48%;  $P < 0.02$ ), an operation on the colon (55% vs 25%;  $P < 0.005$ ), or an operation with a higher wound classification ( $P < 0.02$ ). Mortality was higher in patients with intra-abdominal infection than in those without (44% vs 20%;  $P < 0.02$ ). Wound dehiscence after emergent operations, and operations with a higher wound classification, especially those involving the colon, should raise concern for intra-abdominal infection. Thorough abdominal exploration should be performed at the time of dehiscence repair. Before nonoperative management is chosen, intra-abdominal infection should be excluded.

DESPITE ADVANCES IN surgical technique and postoperative care, the incidence of wound dehiscence after abdominal surgery has remained relatively constant over many years. Historically, abdominal wound dehiscence

was a clear indication for operative repair. Several recent reports, however, advocate nonoperative management of these patients with observation, dressing changes, and late ventral hernia repair.<sup>1-4</sup>

Among the many reported risk factors for wound dehiscence is concurrent infection. In recent years, we have noted a seemingly large number of intra-abdominal infections identified during abdominal exploration at the time of dehiscence repair. Therefore, we reviewed our experience with abdominal wound dehiscence to determine the frequency of concurrent intraabdominal infection, to identify factors that would distinguish patients with abdominal wound dehiscence and intra-abdominal infection from those without intra-abdominal infection, and to determine what impact potential intra-abdominal infection has on management decisions.

## Methods

A retrospective review of all patients with wound dehiscence after abdominal surgery occurring between July 1, 1987 and June 30, 1994 at MetroHealth Medical Center and the Veterans Affairs Medical Center in Cleveland, Ohio was completed. Patients with abdominal wound dehiscence were identified from morbidity and mortality records and discharge ICD-9 coding. Dehiscence was defined as disruption of all layers of the abdominal wall. All patients were greater than 18 years of age. Urologic and gynecologic procedures were excluded from consideration.

Medical records were reviewed for demographic data, medical history, and the presence of known risk factors for dehiscence and postoperative infection. The operative record for the procedure immediately preceding the dehiscence was reviewed to determine the procedure performed, whether the operation was emergent or elective, the American Society of Anesthesiologists (ASA) classification of physical status, the type of incision used, the method of wound closure, and the wound classification. Timing and presentation of the wound dehiscence were determined from progress notes. The clinical status of the patient before the diagnosis of dehiscence, including the presence of fever, leukocytosis, or wound infection, was determined from progress notes and laboratory reports. Finally, management of the wound dehiscence was recorded. For those patients managed nonoperatively, stated reasons for not returning to the operating room for wound closure and any studies used to assist in making this decision were noted. For those patients managed operatively, findings at operation were recorded. Mortality, defined as any death that occurred before discharge from the hospital, was also recorded.

Collected data were analyzed using the Student's t test for continuous variables and Chi-square or Fisher's exact test for noncontinuous variables. A P value of less than 0.05 was considered to be significant. Results

One hundred seven incidents of abdominal wound dehiscence were identified during the study period. There were 85 men and 22 women, with a mean age of 58 years. The operative procedures performed before dehiscence are listed in Table 1. Ninety-three per cent of these operations were performed through a midline incision.

The diagnosis of dehiscence was made between 3 and 23 days postoperatively and occurred most frequently on postoperative day 8. In 67 cases (63%), dehiscence was identified after drainage of serosanguinous fluid from the wound. Evisceration occurred in 18 (17%).

Seventeen patients (16%) were managed nonoperatively. Three were preterminal and died without further intervention. Computed tomography of the abdomen and pelvis was used to exclude intra-abdominal pathology in 6, all of whom had fever and/or leukocytosis. The remainder were clinically well and thought to be at low risk for evisceration. Ninety patients (84%) underwent surgical repair of the abdominal wound. No intra-abdominal pathology was present in 43 patients (40%), although 7 had necrotizing fasciitis. Intra-abdominal infection was diagnosed in 47 patients (44%), including 32 with an abscess and 15 with an anastomotic disruption. Only those patients who were managed operatively were included for further analysis.

No differences could be identified between patients with or without intra-abdominal infection with respect to demographics or comorbid conditions (Table 2). Presentation was similar in both groups. Dehiscence was identified on average on postoperative day 7 (range, 3-17) in those without intra-abdominal infection and on postoperative day 8 (range 3-23) in those with intra-abdominal infection. Patients who were managed nonoperatively tended to present later, on average on postoperative day 11 (range, 6-21;  $P < 0.01$ ). The most common presentation was that of serosanguinous drainage from the wound. Evisceration occurred in 12 patients (28%) without intra-abdominal infection and in 6 patients (13%) with intra-abdominal infection ( $P > 0.05$ ).

Wound infection was diagnosed before dehiscence in 15 patients without intra-abdominal infection and in 24 patients with intra-abdominal infection ( $P > 0.05$ ). Systemic signs of infection did not differ between groups. Average maximum temperature in the 24 hours preceding dehiscence was 37.7°C in the group without intra-abdominal

infection and 38.2°C in the group with intra-abdominal infection ( $P > 0.05$ ). Likewise, leukocyte counts in the 24 hours before reoperation were similar in both groups (14,800/mm<sup>3</sup> vs 16,000/mm<sup>3</sup>,  $P > 0.05$ ).

Several differences in the nature of the initial operation were identified between patients with abdominal wound dehiscence who had intra-abdominal infection and those who did not. Thirty-five of the 47 patients who were found to have intra-abdominal infection had undergone an emergency operation, compared with 20 of 43 who did not have intra-abdominal infection ( $P < 0.02$ ). Wound classification of the initial procedure also differed between the groups (Table 3). Patients with intra-abdominal infection at the time of dehiscence tended to have a higher wound classification for the initial procedure, with 55 per cent having a class III wound, whereas only 26 per cent of patients with dehiscence without intra-abdominal infection had a class III wound ( $P < 0.02$ ). Further examination of the procedures performed before dehiscence revealed a greater frequency of operations on the colon ( $P < 0.004$ ) in the group with intra-abdominal infection compared with those without intra-abdominal infection (Table 4). Intra-abdominal infection was not seen in patients with abdominal wound dehiscence after vascular procedures ( $P < 0.01$ ). No other operative data, including use of preoperative antibiotics, type of incision, length of operation, technique of closure, ASA classification, and transfusion or volume requirements, differed significantly between patients with intra-abdominal infection and those without intra-abdominal infection.

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TABLE  
TABLE 2.

The overall mortality in patients with abdominal wound dehiscence was 35 per cent. However, the mortality in the subgroup of patients with intra-abdominal infection was 44%, compared with 20 per cent in the group without intra-abdominal infection ( $P < 0.02$ ).

## Discussion

The incidence of dehiscence after abdominal surgery is reported to be 0.25 to 3 per cent and has not changed substantially over many years.<sup>1,5-18</sup> The wide range in the reported incidence is the result of differences in the way dehiscence is defined and the types of cases included in the various series. Studies reporting a lower incidence of dehiscence tend to include all abdominal operations, such as appendectomy through a right lower quadrant incision, hernia repair, and gynecologic procedures, all of which have very low rates of dehiscence.

Numerous authors have reviewed their experience with large numbers of patients who have undergone abdominal operations in an attempt to define the etiology of postoperative wound disruption. Although no single etiologic factor can be identified in all cases, a number of factors have been shown to increase the patient's risk for dehiscence. Most authors report a higher incidence of dehiscence with midline incisions, with a higher incidence in upper compared with lower midline incisions.<sup>13-18</sup> Colon operations<sup>8,10,18</sup> and operations for peptic ulcer disease<sup>13,16-18</sup> have been associated with an increased incidence of dehiscence when compared with other intra-abdominal procedures. Some authors report an increased incidence of dehiscence after emergency laparotomy.<sup>5,6,10</sup> Mechanical factors have been implicated in dehiscence including abdominal distension secondary to ileus or obstruction, coughing, retching, and vomiting.<sup>7,11,13,15-18</sup> Patient-related factors that have been associated with an increased incidence of dehiscence include male gender,<sup>6,10-19</sup> increasing age,<sup>5,12,19</sup> malignancy,<sup>7,10,12,16</sup> malnutrition,<sup>7,11,12,18</sup> and other comorbid conditions. Although technical factors are frequently cited as contributing to wound dehiscence, the method of abdominal wound closure and the choice of suture material have not been shown to affect dehiscence rates.

The contribution of infection to the development of dehiscence has been debated. Wound infection has been reported by many to increase the risk of dehiscence,<sup>5, 8, 11,12,15,18,19</sup> whereas others report no association between wound infection and wound dehiscence.<sup>3, 14</sup> However, in most series that include a "control" group, usually consisting of randomly selected patients undergoing similar procedures during the same time period, the incidence of wound infection is substantially greater in those patients who develop dehiscence than in those who do not.<sup>5, 11,12,16, 19</sup> The role of remote infection is less clear. Although many series list "infection" as a contributing factor, few distinguish wound infection from remote infection, and none provide data to support the conclusion that distant infection is a contributing factor.

The frequency of intra-abdominal infection reported in series of abdominal wound dehiscence ranges from 9 to 33 per cent. Harbrecht et al.<sup>2</sup> reported a series of 113 urgent relaparotomies in which 19 patients underwent relaparotomy for abdominal wound dehiscence. Five of these (26%) were found to have an intra-abdominal infection, which the authors described as "presumably causative." In a series published by Greenburg<sup>6</sup> of 32 patients with evisceration over 5 years, 10 were found to have an intra-abdominal abscess. Knight and Griffen<sup>21</sup> reported on 1000 consecutive laparotomies using a specific closure technique. Among the complications reported were three wound dehiscences, including one in a patient with an anastomotic leak. Schiebel and Creech<sup>22</sup> reported 23 patients with abdominal wound dehiscence at a single institution over 10 years, 2 of whom were found to have intra-abdominal infection at the time the abdominal dehiscence was repaired. One patient had a subdiaphragmatic abscess that followed an emergent gastrectomy, and the second had peritonitis after operation for a perforated duodenal ulcer. In a retrospective case-control study, Riou et al.<sup>12</sup> identified septicemia, defined by positive blood cultures, fever, and leukocytosis, as a risk factor for wound dehiscence. In this series, septicemia occurred in 42 per cent of patients with dehiscence compared with only 10 per cent of the controls. In our series, intra-abdominal infection was identified in 44 per cent of patients presenting with abdominal wound dehiscence. Although this incidence is somewhat higher than that previously reported, differences in the types of procedures performed, patient factors, and the focus of the reports may contribute to the discrepancy in infection rates.

| Wound Class | No. Infection (n = 57) | Intra-abdominal Infection (n = 47) |
|-------------|------------------------|------------------------------------|
| I           | 6                      | 2                                  |
| II          | 28                     | 17                                 |
| III         | 11                     | 20                                 |
| IV          | 8                      | 6                                  |

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TABLE 3.

| Procedures             | No. Infection (n = 85) | Intra-abdominal Infection (n = 47) |
|------------------------|------------------------|------------------------------------|
| Colon                  | 11                     | 25                                 |
| Small bowel            | 2                      | 0                                  |
| Gastrointestinal       | 8                      | 0                                  |
| Esophageal             | 0                      | 0                                  |
| Pancreatic             | 1                      | 0                                  |
| Biliary                | 0                      | 0                                  |
| Vascular               | 6                      | 0                                  |
| Cervical               | 0                      | 0                                  |
| Unspecified laparotomy | 2                      | 0                                  |

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TABLE 4.

Impaired wound healing related to remote infection was first reported by Alexis Carrel in 1924.<sup>23</sup> He created cutaneous wounds in dogs, and, after several days of healing, injected turpentine into the dog's flanks to induce abscess formation. Progression of wound healing, specifically epithelialization and contraction, slowed dramatically or halted in the animals with abscesses. Carrel hypothesized the presence of a soluble substance in the serum that mediated the effect of the remote inflammatory process on healing of the cutaneous wound.<sup>23</sup> More recently, de Haan et al.<sup>24</sup> studied the effect of infection on wound healing in a rat model. Infection, in the form of bacteremia or muscle abscess, was found to decrease bursting strength of both gastric and abdominal wounds. Sterile muscle abscesses, induced by turpentine injection, were also shown to decrease wound-bursting strength. Drainage of the abscess just after wounding resulted in woundbursting strength equivalent to those of animals without infection. Although the mechanisms whereby wound healing is impaired by remote infection have not been defined, recent studies on the healing of colonic anastomoses suggest that sepsis impedes colonic healing by inhibiting collagen synthesis at the anastomotic site.<sup>25</sup> The specific mediators of this effect on collagen synthesis have not yet been identified. A similar process affecting abdominal wounds seems likely. Thus, remote infection appears to have the capacity to impair wound healing, and, as a consequence, increase the risk for dehiscence. Other sites of postoperative infection are likely to have similar detrimental effects on wound healing, although such infections were not considered in this study.

Because of the high incidence of intra-abdominal infection observed in our patients with abdominal wound dehiscence, an analysis of potential factors predictive of concurrent intra-abdominal infection was completed. Traditional indicators of systemic infection, such as fever and leukocytosis, did not distinguish patients with dehiscence and intraabdominal infection from those without intra-abdominal infection. Factors found to be associated with intra-abdominal infection in patients with wound dehiscence included higher wound classification, emergency operations, and operations on the colon.

Wound classification clearly correlates with the occurrence of wound infection after surgery. The wound infection rate for dirty wounds is 38.3 per cent compared with 1.8 per cent for clean wounds.<sup>26</sup> Wound infection was present in 51 per cent of patients with intra-abdominal infection and 35 per cent of those without intra-abdominal infection, and, when stratified by wound class, wound infection rates did not differ between patients with intraabdominal infection and those without infection. Although there are no data available correlating intra-abdominal infection with wound classification, it is not surprising that patients undergoing procedures with higher wound classes would be more likely to have intra-abdominal infection at the time of dehiscence.

Emergency operation has been associated with both an increased risk of abdominal wound infection<sup>27</sup> and abdominal wound dehiscence.<sup>5</sup> 10 Factors that might contribute to the increased risk for infection after emergency operations include extensive blood loss and tissue damage that may accompany operations for trauma, gross peritoneal contamination, which is often encountered in emergency situations, and the lack of preoperative bowel preparation. The marked preponderance of colonic procedures in the group of patients with abdominal wound dehiscence and intra-abdominal infection cannot readily be explained. Management of wound dehiscence has traditionally consisted of operative repair. Most series include a number of patients who were not operated for various reasons, most frequently because of poor medical status. However, several recent reports advocate nonoperative management with late ventral hernia repair.<sup>1-4</sup> In practice, nonoperative management seems to be relatively frequent in our hospitals, having been used in 16 per cent of cases in this series. A nonoperative approach was selected based on the patient's clinical condition; those not expected to survive were managed nonoperatively, as were those the surgeon felt to be clinically well and at low risk for evisceration. Although the presence or absence of fever or leukocytosis did not distinguish patients with dehiscence and concomitant intra-abdominal infection from those without intraabdominal infection, they were frequently cited by clinicians in the decision to obtain computed tomography of the abdomen and pelvis to rule out intraabdominal infection when nonoperative management was contemplated.

Mortality for all patients with dehiscence was 35 per cent, consistent with the 15 to 44 per cent mortality reported in the literature.<sup>11,12,15-18</sup> The high mortality rate accompanying abdominal wound dehiscence is felt by many to reflect the poor medical condition of those patients in whom dehiscence often occurs. The presence of concurrent intra-abdominal infection had a significant impact on mortality. While mortality among patients with dehiscence without intra-abdominal infection was high, at 20 per cent, the presence of concurrent intraabdominal infection resulted in a mortality rate of 44 per cent, more than double that seen in patients without intra-abdominal infection.

Intra-abdominal infection is frequent among patients who present with abdominal wound dehiscence and results in a significant increase in mortality. Standard indicators of systemic infection, specifically fever and leukocytosis, do not differentiate patients with intra-abdominal infection from those without intraabdominal infection. Intra-abdominal infection should be suspected when dehiscence complicates an emergency operation, a colectomy, or a procedure with a high wound classification. At the time of operative repair, the abdomen should be thoroughly explored and any anastomoses or suture lines carefully inspected. If nonoperative management of the dehiscence is considered, evaluation of the abdomen and pelvis using computed tomography is recommended to exclude intra-abdominal infection as a contributing factor in the development of abdominal wound dehiscence.

## DISCUSSION

DR. CHARLES LUCAS (Detroit, Michigan): Dr. Graham and her colleagues have told us about the power of the physiologic warfare that occurs at the microscopic level with the polymorphonuclear neutrophils and their allied helper cells fighting with bacteria in an engagement which has awful killing. The mutual killing of friend and foe alike includes innocent bystanders such as the fibroblasts, which are no longer able to make the elastic or collagen, and the other intracellular substances which promote crosslinking and polymerization. More importantly, the warring cells release substances such as collagenase and elastase, which destroy the existing structures and melt them into oblivion. The effects are global so that the wound dehisces, even though the struggle is taking place far away. Dr. Graham reminds us that technical problems with the colon anastomosis will lead to wound disruption, even though the wound was technically repaired beautifully. One could presume, therefore, that there are some patients with dehiscence and unrecognized intraperitoneal infection, because the friends won the struggle which occurred prior to the wound dehiscence. Like in warfare, prevention is the key. Dr. Graham, have you looked at the techniques for your colon repairs and have you compared the handsewn versus the stapled anastomosis? Some of us "old farts" still use the handsewn anastomosis for doing colon surgery. You talk about highrisk wounds. What was the incidence of primary versus secondary intent closure in these wounds? Hopefully, the high-risk wounds were all treated by second intent. Were retention sutures used in these patients and did they cause any problems with fistulae? When you operate upon patients with necrotizing fasciitis requiring resection, how do you reconstruct? I have one disagreement with the manuscript, and it is only a minor disagreement. They emphasize full exploration

when they take a patient back with wound dehiscence. One has to be careful that full exploration does not lead to a bacteremic shower when an area of abscess can be drained by a lesser technique.

DR. HOMER M. SMATHERS (Detroit, Michigan): I didn't hear the author comment about the type of incision. Many years ago at Receiving Hospital, I reviewed over 400 perforated ulcers and found that 19 per cent of the oblique incisions resulted in infection and 19 per cent of the midline incisions resulted in infection. However, there was not one dehiscence in the oblique incision, and I think this is a major factor which is overlooked.

DR. GRAHAM: I will address the last question first. We did look at the type of incision and over 93 per cent of the procedures were performed through a midline incision so it is very difficult to compare. In terms of the technique for the colon repairs, there were a mix of handsewn and stapled anastomoses, and we didn't look at the association with intra-abdominal infection or association with dehiscence. Primary and secondary intent closures were based on the findings of the initial procedure and, again, we didn't look at that in relation as to whether or not those wounds dehiscenced. In the patients who were closed with retention sutures, there were no reports of fistulae among those patients. Finally, in the patients with necrotizing fasciitis, some were repaired with mesh; most were debrided and eventually skin-grafted.

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